Prior Heavy Exercise Enhances Performance during Subsequent Perimaximal Exercise

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ABSTRACT

JONES, A. M., D. P. WILKERSON, M. BURNLEY, and K. KOPPO. Prior Heavy Exercise Enhances Performance during Subsequent Perimaximal Exercise. *Med. Sci. Sports Exerc.*, Vol. 35, No. 12, pp. 2085–2092, 2003. **Purpose:** To test the hypothesis that prior heavy exercise increases the time to exhaustion during subsequent perimaximal exercise. **Methods:** Seven healthy males (mean ± SD 27 ± 3 yr; 78.4 ± 0.7 kg) completed square-wave transitions from unloaded cycling to work rates equivalent to 100, 110, and 120% of the work rate at $\dot{V}O_{2peak}$ (W-$\dot{V}O_{2peak}$) after no prior exercise (control, C) and 10 min after a 6-min bout of heavy exercise at 50% Δ (HE; half-way between the gas exchange threshold (GET) and $\dot{V}O_{2peak}$), in a counterbalanced design. **Results:** Blood [lactate] was significantly elevated before the onset of the perimaximal exercise bouts after prior HE (~2.5 vs ~1.1 mM; $P < 0.05$). Prior HE increased time to exhaustion at 100% (mean ± SEM. C: 386 ± 92 vs HE: 613 ± 161 s), 110% (C: 218 ± 26 vs HE: 284 ± 47 s), and 120% (C: 139 ± 18 vs HE: 180 ± 29 s) of W-$\dot{V}O_{2peak}$, (all $P < 0.01$). $\dot{V}O_2$ was significantly higher at 1 min into exercise after prior HE at 110% W-$\dot{V}O_{2peak}$ (C: 3.11 ± 0.14 vs HE: 3.42 ± 0.16 L·min^{-1}; $P < 0.05$), and at 1 min into exercise (C: 3.25 ± 0.12 vs HE: 3.67 ± 0.15; $P < 0.01$) and at exhaustion (C: 3.60 ± 0.08 vs HE: 3.95 ± 0.12 L·min^{-1}; $P < 0.01$) at 120% of W-$\dot{V}O_{2peak}$. **Conclusions:** This study demonstrate that prior HE, which caused a significant elevation of blood [lactate], resulted in an increased time to exhaustion during subsequent perimaximal exercise presumably by enabling a greater aerobic contribution to the energy requirement of exercise. **Key Words:** TIME TO EXHAUSTION, $\dot{V}O_2$ KINETICS, WARM-UP, FATIGUE

Gerbino et al. (14) demonstrated that the performance of heavy intensity exercise (i.e., exercise above the gas exchange threshold; GET), but not moderate-intensity exercise (<GET), speeded the overall pulmonary oxygen uptake ($\dot{V}O_2$) kinetics during subsequent heavy-intensity exercise. This speeding of the overall $\dot{V}O_2$ response was shown subsequently to result from a reduction in the amplitude of the $\dot{V}O_2$ "slow component" with no change in the time constant of the fundamental $\dot{V}O_2$ response in phase II (10,20). In studies in which sufficient time was allowed between exercise bouts to restore baseline $\dot{V}O_2$, prior heavy-intensity exercise resulted in an increased amplitude of the fundamental response and a reduction in the amplitude of the $\dot{V}O_2$ slow component with a similar end-exercise $\dot{V}O_2$ at 6–8 min (3,7,8). This altered $\dot{V}O_2$ response was associated with a reduction in the accumulation of blood [lactate] over the exercise bout, an attenuated $\dot{V}CO_2$ response, and a reduced overall O_2 deficit. In keeping with these observations, measurements of muscle blood flow, arterial-femoral venous O_2 difference, and the changes in and release of muscle metabolites indicate an increased oxidative contribution to energy metabolism and a reduction in substrate level phosphorylation in the second of two high-intensity exercise bouts with no change in total ATP turnover (1,23). A recent magnetic resonance spectroscopy study has confirmed that prior heavy-intensity exercise results in a significant sparing of intramuscular [PCr] during subsequent heavy intensity exercise (29).

There is consensus, therefore, that prior heavy-intensity exercise reduces the overall O_2 deficit, limits the depletion of intramuscular [PCr] and the production of lactic acid, and attenuates the $\dot{V}O_2$ slow component during subsequent heavy exercise. These effects would be expected to increase exercise tolerance (14,17,28) or at least to allow a better tolerance of exercise than might be expected for the prevailing metabolic conditions (14). The purpose of this study was therefore to test the hypothesis that prior heavy-intensity exercise would extend the time to exhaustion during subsequent perimaximal exercise.

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METHODS

Participants. Seven healthy males (mean ± SD 27 ± 3 yr; 78.4 ± 0.7 kg) volunteered to participate in this study that was approved by the Manchester Metropolitan University ethics committee. All participants gave their written informed consent after the benefits and risks of participation in the study had been explained to them. The participants were regularly active in recreational exercise but were not highly trained.

Experimental design. The participants visited the laboratory on seven occasions over a 3-wk period. The first visit was used to determine the $\dot{V}O_{2peak}$ and GET, and the other visits were used to complete the experimentation. On a given day, participants exercised to exhaustion at either 100, 110, or 120% of the work rate at $\dot{V}O_{2peak}$ (see below), after either no prior exercise (control condition) or after prior heavy-intensity exercise.

The experimental conditions were presented to the participants in a counterbalanced design. All participants reported to the laboratory rested (having performed no strenuous activity in the preceding 24-h), well hydrated, and having abstained from food, alcohol, and caffeine in the 3-h before testing. Tests were conducted in an air-conditioned laboratory (temperature 19°C) at the same time of day for each participant (± 2 h) and were separated by at least 24 h.

Measurement of GET and $\dot{V}O_{2peak}$. All tests were performed on an electronically braked cycle ergometer (Ergoline, Jaeger), which controlled external work rate independent of pedal cadence. Participants self-selected a cadence for the $\dot{V}O_{2peak}$ test (this ranged between 80 and 90 rpm). Although it is known that cadence can influence time to exhaustion, this was controlled for in the present study because all exercise tests were performed at the same cadence (± 2 rpm for any given subject). The $\dot{V}O_{2peak}$ test began with 3 min of baseline cycling at 20 W (the lowest work rate available on the ergometer), after which work rate was increased by 30 $W \cdot min^{-1}$ until volitional exhaustion (reached in 9–11 min). Throughout the test, pulmonary gas exchange was measured breath-by-breath as described below. The $\dot{V}O_{2peak}$ was determined as the highest value recorded in any 30-s period before the participant's volitional termination of the test. The GET was determined as the first disproportionate increase in $\dot{V}CO_2$ from visual inspection of individual plots of $\dot{V}CO_2$ versus $\dot{V}O_2$ by an experienced reviewer. The work rate at $\dot{V}O_{2peak}$ (W-$\dot{V}O_{2peak}$) was defined as the work rate at which $\dot{V}O_2$ began to plateau near the end of the test ($N = 5$) or reached its highest value when no plateau was evident ($N = 2$). For each individual, work rates equivalent to 50% Δ (half-way between the work rate at the gas exchange threshold (GET) and W-$\dot{V}O_{2peak}$; heavy-intensity exercise), and 100, 110, and 120% of the W-$\dot{V}O_{2peak}$ (perimaximal exercise) were calculated.

Experimental tests. Participants were required to maintain a low (100% W-$\dot{V}O_{2peak}$), medium (110% W-$\dot{V}O_{2peak}$), or high (120% W-$\dot{V}O_{2peak}$) perimaximal work rate for as long as possible. These exhaustive bouts were preceded either by no prior exercise (control condition) or by heavy-intensity prior exercise. The participants were not aware of the purpose of the study, and at no point during or after any test was information given regarding performance time.

The prior exercise conditions involved participants performing 3 min of baseline cycling at 20 W, followed by a square-wave transition to a work rate requiring 50% Δ; this work rate was maintained for 6 min. After the prior exercise bout, the participants rested for 7 min before cycling at 20 W for a further 3 min. The work rate was then increased abruptly to the predetermined perimaximal work rate (100, 110, or 120% of W-$\dot{V}O_{2peak}$), and participants attempted to maintain this work rate for as long as possible. The exhaustive trial was terminated when the self-selected cadence dropped by >5 rpm. Time to exhaustion was recorded to the nearest second. The perimaximal bouts performed in the control condition were identical except that they were not preceded by heavy-intensity exercise. Critical power (CP) and anaerobic work capacity (W') (28) were calculated from the three perimaximal exercise bouts performed in each condition using the linear model of work rate versus 1/time.

Fingertip blood samples (~25 μL) were collected into a capillary tube immediately before and immediately after the perimaximal exercise bouts. Blood samples were subsequently analyzed for blood [lactate] using an automated lactate analyser (YSI stat 2300, Yellow Springs, OH). This analyzer was calibrated before each test with a 5-mM lactate standard supplied by the manufacturer (YSI 2747).

Measurement of pulmonary gas exchange and heart rate. Pulmonary gas exchange was measured breath by breath during all exercise tests. Participants breathed through a low-resistance volume transducer (Jaeger Triple V, Hoechberg, Germany), which had a dead space of 90 mL. Gas was continuously drawn down a capillary line into rapid-response gas analyzers (Jaeger Oxycon Alpha). Gas exchange variables were calculated and displayed breath by breath once the delay between the volume and concentration signals had been accounted for. The volume transducer was calibrated before each test with a 3-L calibration syringe, and the analyzers were calibrated with gases of known concentration. Heart rate was recorded every 5-s by using short range telemetry (Polar PE 4000, Kempele, Finland).

Baseline $\dot{V}O_2$ was defined as the average of the $\dot{V}O_2$ values measured between the second and third minutes of exercise at 20 W before the transitions to the perimaximal work rates. The $\dot{V}O_2$ at 1 min of the exhaustive bouts was calculated from the average $\dot{V}O_2$ of a 30-s period centred on 1 min. The end $\dot{V}O_2$ was defined as the average of the $\dot{V}O_2$ values measured during the last 30 s of exercise. The response curves of $\dot{V}O_2$ were also described using mathematical modeling procedures. The $\dot{V}O_2$ response to supra-maximal exercise (110 and 120% W-$\dot{V}O_{2peak}$) was expected to conform to a mono-exponential function after the cardiodynamic phase, whereas the response to exercise at ~100% W-$\dot{V}O_{2peak}$ might have been complicated by the emergence of the $\dot{V}O_2$ slow component (26). To allow a comparison of the rate of $\dot{V}O_2$ response after the onset of exercise, the data

TABLE 1. Time to fatigue, blood [lactate], and heart rate values during the perimaximal exercise bouts.

	100% W – $\dot{V}O_{2peak}$	110% W – $\dot{V}O_{2peak}$	120% W – $\dot{V}O_{2peak}$
Time to fatigue (s)			
C	386 ± 92	218 ± 26	139 ± 18
HIPE	613 ± 161†	284 ± 47†	180 ± 29†
[Lactate] start (mM)			
C	1.1 ± 0.1	1.1 ± 0.1	1.2 ± 0.1
HIPE	2.4 ± 0.3†	2.5 ± 0.4†	2.6 ± 0.5†
[Lactate] end (mM)			
C	7.2 ± 0.7	7.0 ± 0.7	6.0 ± 0.7
HIPE	7.2 ± 0.4	8.6 ± 0.4	6.9 ± 0.8
Δ [Lactate] (mM)			
C	6.1 ± 0.7	5.9 ± 0.6	4.8 ± 0.7
HIPE	4.8 ± 0.4	6.1 ± 0.4	4.3 ± 0.6
HR start (beats·min^{-1})			
C	92 ± 3	88 ± 4	89 ± 5
HIPE	103 ± 3†	101 ± 3†	102 ± 3†
HR end (beats·min^{-1})			
C	177 ± 3	174 ± 3	167 ± 3
HIPE	180 ± 1	178 ± 3†	178 ± 3†

C, control; HIPE, heavy-intensity prior exercise. Values are means ± SEM.
† Significant difference ($P < 0.05$) between C and HIPE at corresponding intensity.

were modeled using both single and double exponential models in order to isolate the $\dot{V}O_2$ fast component response. The initial cardiodynamic component was ignored by eliminating the first 20 s of data after the onset of exercise. The models used can be expressed as follows:

$$\dot{V}O_2(t) = \dot{V}O_{2\,baseline} + A_p(1 - e^{-(t - Tdp)/\tau p}) \quad [1]$$

$$\dot{V}O_2(t) = \dot{V}O_{2\,baseline} + A_p(1 - e^{-(t - Td_p)/\tau_p}) + A_s(1 - e^{-(t - Td_s)/\tau_s}) \quad [2]$$

The exponential models included amplitudes (A_p and A_s), time constants (τ_p and τ_s), and delay times (Td_p and Td_s). A_p, τ_p, and Td_p describe the $\dot{V}O_2$ fundamental component, while A_s, τ_s, and Td_s describe the $\dot{V}O_2$ slow component. A nonlinear least-square algorithm was used to determine the parameters of the best fitting curve. The mean-square amplitudes were used to evaluate whether the monoexponential or biexponential model provided the best fit to each data set. Owing to concerns over the relatively low signal/noise ratio with only one exercise transition in each condition, we chose to report only the mean response time for the primary phase of the response ($TD_p + \tau_p$).

Statistical analysis. Differences in time to exhaustion and physiological response between the perimaximal exercise bouts preceded by no prior exercise and those preceded by heavy-intensity prior exercise were tested for statistical significance using paired *t*-tests. Statistical significance was set at $P < 0.05$.

RESULTS

The mean (± SD) $\dot{V}O_{2peak}$ of the participants was 50.3 ± 3.3 mL·kg^{-1}·min^{-1}, with GET occurring at 52 ± 12% of $\dot{V}O_{2peak}$. Heavy-intensity prior exercise was performed at 253 ± 51 W ($N = 7$). The work rates used in the exhaustive bouts were 322 ± 39, 357 ± 43, and 392 ± 48 W for the 100, 110, and 120% W-$\dot{V}O_{2peak}$ conditions, respectively.

Time to exhaustion was significantly longer in all of the perimaximal bouts after heavy-intensity prior exercise compared with the control condition (Table 1 and Fig. 1). The CP was not significantly affected by the performance of prior high-intensity exercise (control: 284 ± 35 vs heavy exercise: 289 ± 26 W), but the increase in W' approached statistical significance (control: 15.5 ± 2.2 vs heavy exercise: 18.2 ± 3.5 kJ; $P = 0.06$), (Fig. 2). Heavy-intensity prior exercise caused a significant increase in blood [lactate], which was still present at the onset of the perimaximal exercise bouts (Table 1). However, no significant differences in blood [lactate] were observed at the termination of the perimaximal exercise bouts. Heart rate immediately before the onset of the perimaximal exercise bouts was significantly higher after prior heavy-intensity exercise compared with control (Table 1). This higher heart rate persisted throughout exercise and was still evident at the cessation of the exhaustive bouts, reaching significance at 110% and 120% W-$\dot{V}O_{2peak}$ ($P < 0.05$).

Table 2 shows the $\dot{V}O_2$ responses to perimaximal exercise after no prior exercise and after heavy-intensity prior exercise. There was no significant difference in baseline $\dot{V}O_2$ at the onset of the perimaximal exercise bouts between the control and prior heavy-intensity exercise conditions. The $\dot{V}O_2$ kinetics in the primary adaptive phase (as given by the mean response time) were not significantly altered by the performance of prior heavy-intensity exercise. At 100% W-$\dot{V}O_{2peak}$, there was no significant difference in the $\dot{V}O_2$ at 1 min into exercise or at exhaustion. At 110% W-$\dot{V}O_{2peak}$, $\dot{V}O_2$ at 1 min into exercise was significantly higher than in the control condition ($P < 0.05$), but there was no significant difference in the $\dot{V}O_2$ at exhaustion. At 120% W-$\dot{V}O_{2peak}$, $\dot{V}O_2$ was significantly higher both at 1 min into exercise ($P < 0.05$) and at exhaustion ($P < 0.05$) compared with the control condition.

DISCUSSION

Consistent with our hypothesis, the results indicate that prior heavy-intensity exercise leads to a significant increase in time to exhaustion during subsequent perimaximal exercise. Previous research has demonstrated that prior high-intensity exercise results in an increased oxidative contribution to energy metabolism during subsequent high-intensity exercise, with a corresponding reduction in the oxygen deficit, as reflected in a reduced rate of substrate-level phosphorylation (1,6,23,24,29). There is direct evidence that muscle blood flow and O_2 extraction are increased during high-intensity cycle exercise when it is preceded by a high-intensity warm-up (1,23). Furthermore, Rossiter et al. (29) reported that prior heavy leg extension exercise resulted in a 9% sparing of [PCr] over the first 2–3 min of an identical exercise bout performed 6 min later. Other groups have also shown that total anaerobic energy turnover is substantially reduced (by 25–45%) in the second of two high-intensity exercise bouts (1,11,23). The reduced lactate production and sparing of intramuscular [PCr] resulting from the increased muscle aerobic energy turnover might be expected to predispose to increased exercise tolerance (14), as indeed we found in the present study. This is

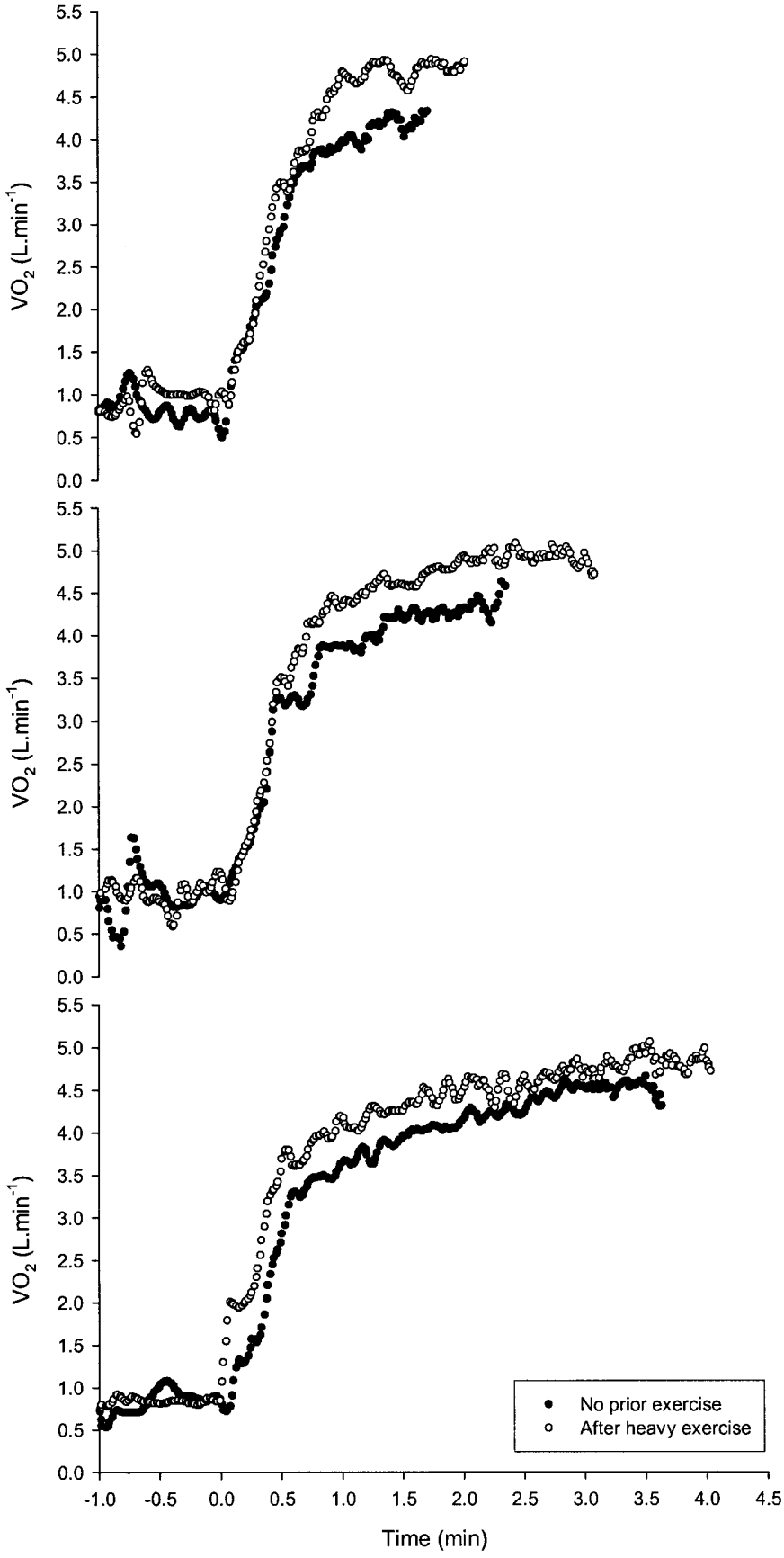

FIGURE 1—Breath-by-breath $\dot{V}O_2$ response in one participant who performed exercise at 100% (lower panel), 110% (center panel), and 120% (upper panel) W-$\dot{V}O_{2peak}$ with no prior exercise (control; *closed circles*) and prior heavy-intensity exercise (*open circles*). Note the elevated $\dot{V}O_2$ throughout exercise after prior heavy-intensity exercise.

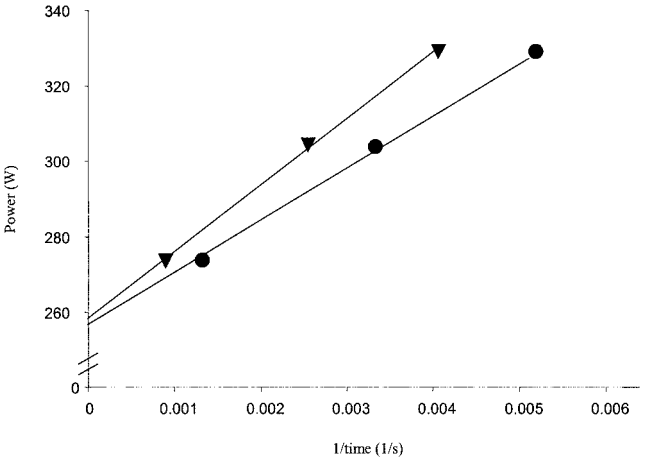

FIGURE 2—Altered power-time relationship in a typical subject after the performance of prior heavy-intensity exercise. Data are presented as power output vs 1/time. Note the change in the slope of the line (indicating an increased W') but the similar y intercept (indicating a similar CP).

because the point of exhaustion is often presumed to coincide with a depletion of intra-muscular phosphates to some critical value and/or the attainment of a critically low intracellular pH or high [lactate] (but see below) (12,15,27,28).

In the present study, the performance of prior heavy-intensity exercise led to a dramatic increase in the time to exhaustion during subsequent perimaximal exercise (29–59% compared with the control condition). Interestingly, however, prior heavy-intensity exercise did not cause a significant improvement in the CP but did influence the curvature constant of the power-time relationship, resulting in an increased W' that approached statistical significance. It is likely that the difference in W' would have reached statistical significance if our subject sample had been slightly larger, indicating that this is a real effect that is worthy of further investigation. The W' is considered to represent a finite energy reserve comprising O_2 stores (note that this would be relatively small), high-energy phosphates, and energy derived through anaerobic glycolysis (28). It is unclear, therefore, how the performance of heavy exercise might enhance the derivation of energy through predominantly O_2-independent pathways (i.e., increase the "anaerobic capacity") unless the effect of prior heavy-intensity exercise is to retard the rate at which metabolites that ultimately result in fatigue accumulate during subsequent exercise (see below).

TABLE 2. Oxygen uptake response to the perimaximal exercise bouts.

	100% W – $\dot{V}O_{2peak}$	110% W – $\dot{V}O_{2peak}$	120% W – $\dot{V}O_{2peak}$
Baseline $\dot{V}O_2$ (mL·min^{-1})			
C	868 ± 52	859 ± 28	834 ± 55
HIPE	840 ± 50	799 ± 30	866 ± 31
1st minute $\dot{V}O_2$ (mL·min^{-1})			
C	2909 ± 112	2865 ± 159	3058 ± 126
HIPE	2953 ± 134	3128 ± 139†	3332 ± 172†
End $\dot{V}O_2$ (mL·min^{-1})			
C	3819 ± 89	3882 ± 124	3524 ± 75
HIPE	3785 ± 78	3844 ± 146	3863 ± 99†
Mean response time (s)			
C	40.7 ± 3.5	39.8 ± 5.2	42.4 ± 8.0
HIPE	36.6 ± 3.9	33.0 ± 4.3	40.0 ± 5.4
End $\dot{V}O_2$ (% of $\dot{V}O_{2peak}$)			
C	95.4 ± 1.8	97.3 ± 2.8	89.7 ± 2.9
HIPE	95.5 ± 1.6	96.3 ± 1.8	95.2 ± 2.0†

C, control; HIPE, heavy intensity prior exercise. Values are means ± SEM.
† Significant difference ($P < 0.05$) between C and HIPE at corresponding intensity.

It is important to point out that the performance of prior moderate-intensity exercise (i.e., "warm-up") does not alter the $\dot{V}O_2$ or blood [lactate] response to subsequent exercise (10,14). Furthermore, moderate-intensity warm-up exercise does not appear to enhance performance during subsequent high-intensity endurance exercise (5,21). We have recently found that ~12 min of prior moderate-intensity exercise, that would have resulted in a similar total energy expenditure to the prior heavy-intensity exercise bout performed in the present study, had a relatively small effect (+2–5%) on the time to exhaustion in subsequent perimaximal exercise bouts (Jones et al., unpublished observations, 2003). It has also been demonstrated that the elevation of muscle temperature by 2–3°C using hot packs or water baths (9,19,22) does not appreciably alter the $\dot{V}O_2$ or blood [lactate] response to subsequent exercise. Collectively, these results suggest that the extended time to exhaustion in the perimaximal bouts after prior heavy-intensity exercise was unlikely to be contingent on an increased muscle temperature.

Blood [lactate] was significantly elevated at the onset of the perimaximal exercise trials after prior heavy-intensity exercise (~2.5 mM), and it might appear paradoxical that time to exhaustion was extended in the face of this residual acidosis. Both the lactate ion and the hydrogen ion have been shown to reduce force production in isolated muscle (15,27), and the accumulation of lactate and the reduction of pH during heavy exercise in humans is believed by many to be a major cause of muscle fatigue (13). However, many of the early studies that led to this view were conducted at unphysiologically low temperatures (~17°C), and when these experiments are repeated at 37°C, the inhibitory effect of intracellular acidification on muscle contractility disappears (30). In an important recent study, Nielsen et al. (25) reported that the addition of 20-mM lactic acid to an isolated muscle preparation almost completely reversed the 75% loss of tetanic force caused by preincubation at an extra-cellular [K^+] of 11 mM. These data indicate that, rather than causing fatigue, acidosis might protect against fatigue resulting from the loss of muscle K^+ during exercise, at least in some situations. In the present study, it is therefore possible that the residual acidosis after prior heavy-intensity exercise served to preserve muscle function and retard the rate at which fatigue developed during the subsequent high-intensity exercise bouts.

It appears that the extent of the metabolic acidosis caused by prior exercise is important in determining the potential for performance enhancement (2,16,18). For example, Karlsson et al. (18) reported that prior exhaustive arm exercise that increased blood [lactate] to ~10 mM resulted in a 31% reduction in the time to exhaustion in exhaustive leg cycle exercise performed 6 min later. More recently, Koppo

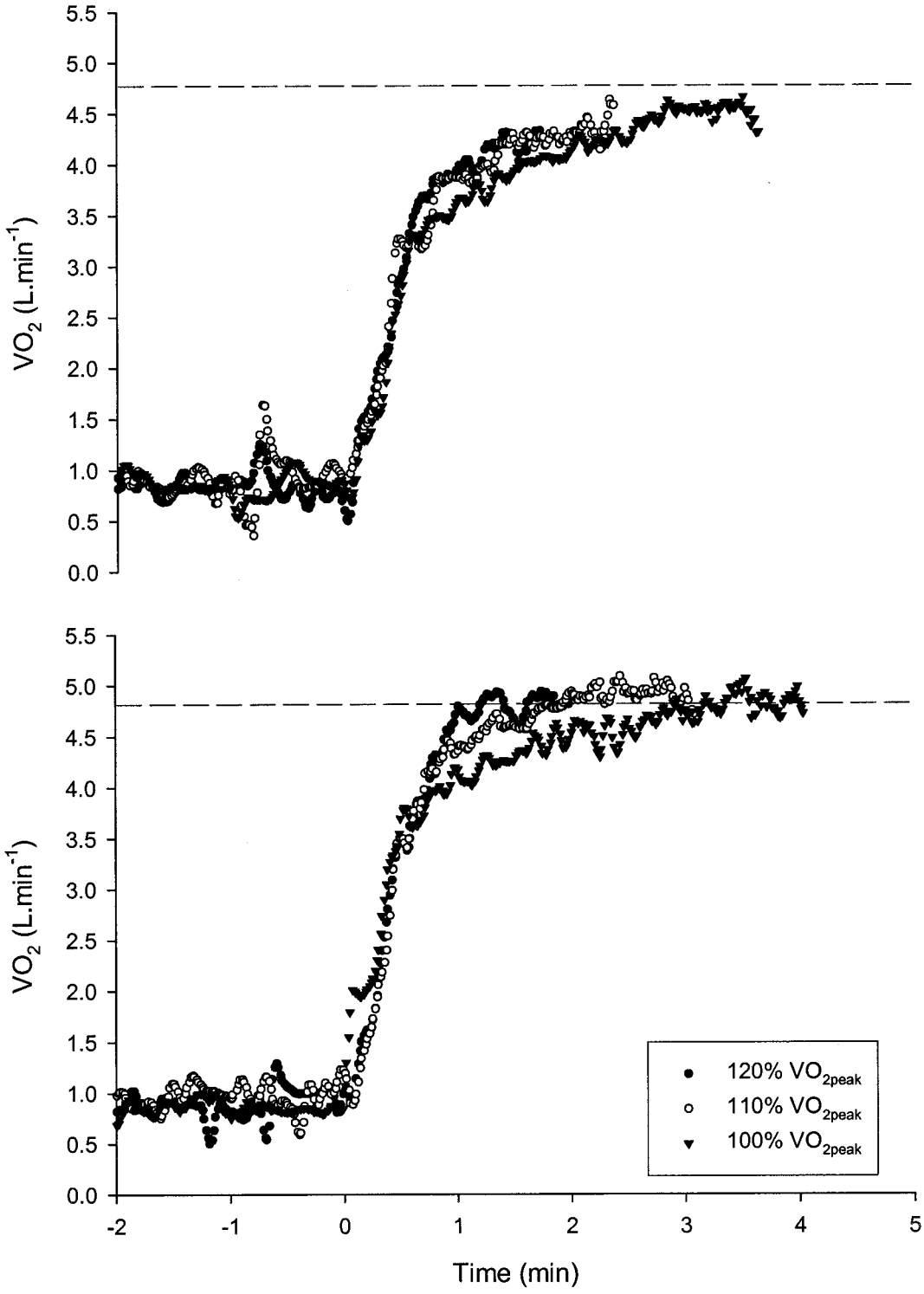

FIGURE 3—Breath-by-breath $\dot{V}O_2$ response to perimaximal exercise without prior exercise (upper panel) and with prior heavy-intensity exercise (lower panel) in a typical participant. Note that in the control condition, exhaustion occurs before $\dot{V}O_{2peak}$ is attained (*dashed horizontal line*). However, prior heavy-intensity exercise results in the attainment of $\dot{V}O_{2peak}$ at all three perimaximal work rates.

and Bouckaert (21) reported that prior exercise at 90% $\dot{V}O_{2peak}$ (as compared with ~75% $\dot{V}O_{2peak}$ in the present study), which resulted in a blood [lactate] of ~6 mM, did not significantly alter time to exhaustion during subsequent exercise at 95% $\dot{V}O_{2peak}$. Also, Bishop et al. (5) reported that a continuous heavy-intensity warm-up at ~75% $\dot{V}O_{2peak}$, which elevated blood [lactate] to ~5.1 mM, did not improve performance in a 2-min all-out kayak ergometer test. It is possible that very-high-intensity prior exercise results in the accumulation of other fatiguing metabolites (e.g., P_i, $H_2PO_4^-$, ammonia) in muscle (30), or that more time is subsequently required for the restoration of extracellular $[K^+]$ (2). Therefore, differences in the rate of lactate clearance compared with the rate of clearance of other "fatiguing" metabolites and the speed with which the electrochemical gradient across the muscle membrane is restored may be important in determining the potential for performance enhancement. It may be that there is an optimal

blood [lactate] of ~2–3 mM that is required for performance enhancement because prior exercise that either results in no change in blood [lactate], or prior exercise and recovery combinations that result in large elevations in blood [lactate] of greater than ~5 mM, does not enhance performance (5,18,21). Moderate-intensity warm-up exercise does not enhance the aerobic contribution to subsequent exercise (10,14), whereas severe-intensity warm-up exercise may not allow sufficient time for restoration of intramuscular high-energy phosphates and/or removal of fatiguing metabolites (e.g., P_i, H^+, $H_2PO_4^-$, or extra-cellular K^+) before the commencement of the criterion exercise challenge.

Although somewhat controversial, it appears that priming exercise does not result in a speeding of the $\dot{V}O_2$ kinetics over the primary adaptive phase of the response during subsequent cycle exercise but that $\dot{V}O_2$ projects to a greater initial amplitude with an unchanged time constant (3,7,8). In the present study, the overall primary $\dot{V}O_2$ kinetics expressed as the mean response time were not significantly affected by prior exercise; rather, $\dot{V}O_2$ appeared to project to and/or reach a higher $\dot{V}O_2$ in the perimaximal exercise bouts with a similar rate of response. Indeed, at 120% W-$\dot{V}O_{2peak}$, the increased time to exhaustion resulting from the performance of prior heavy-intensity exercise allowed $\dot{V}O_{2peak}$ to be attained, whereas in the control condition exercise was terminated before $\dot{V}O_{2peak}$ was reached (Fig. 3). As mentioned above, this suggests that the mechanism(s) causing fatigue in the control condition were in some way attenuated by the performance of prior high-intensity exercise.

The mechanisms responsible for the elevated $\dot{V}O_2$ throughout the transient phase of heavy exercise when it is preceded by a bout of heavy exercise are still debated, but these might feasibly include changes in O_2 availability, enzyme activity, substrate availability, or motor unit recruitment (4,7,11,14). Gerbino et al. (14) suggested that the residual acidosis after a priming bout of heavy exercise might increase muscle vasodilation and result in more homogeneous muscle perfusion at exercise onset. For perimaximal exercise, at least, reduction of any heterogeneity in oxygen delivery to working muscle would be expected to increase $\dot{V}O_2$ and diminish Δ [lactate]. Alternatively, it is possible that changes in putative controllers of muscle respiration such as the phosphorylation potential, [PCr], NADH availability, and cytosolic [Ca^{2+}] after prior heavy exercise might reduce any inherent inertia in the key enzymes controlling oxidative metabolism and result in either a reduced "lag" before muscle O_2 consumption increases appreciably after the onset of subsequent exercise (4) or a more rapid adjustment to the "steady state" requirement. It is also known that prior exercise results in a stockpiling of acetyl groups, and it has been suggested that this greater availability of metabolic substrate might enhance mitochondrial ATP production (11). Finally, it has been suggested that the increased $\dot{V}O_2$ might be related to increased motor unit recruitment at the onset of exercise. Burnley et al. (7) reported that muscle iEMG was 19% higher at the onset of the second of two bouts of severe-intensity exercise and noted that the iEMG responses were qualitatively similar to the pulmonary $\dot{V}O_2$ responses in both bouts. Increased motor unit recruitment at the onset of heavy constant work rate exercise might be beneficial to performance because the metabolic strain on each fiber would be reduced.

In conclusion, the performance of prior high-intensity exercise resulted in a significant increase in time to exhaustion during subsequent perimaximal exercise at 100%, 110%, and 120% W-$\dot{V}O_{2peak}$ despite, or perhaps because of, a significant elevation of blood [lactate] (to ~2.5 mM) at exercise onset. The results of this study may have important implications for the preparation of athletes for competition and for the improvement of exercise tolerance in the elderly and in patient populations. The characteristics of the prior exercise intensity, exercise duration, exercise mode, and recovery period that optimize the ergogenic effect of prior heavy-intensity exercise remain to be determined. Elucidation of these characteristics, and examination of the effect of prior heavy-intensity exercise on performance both in athletes and in populations with compromised exercise tolerance, may be fruitful topics for future research.

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